

DEATH CERTIFICATES: check every death certificate attached to C.A.A. over the past two year period, the past five year period; then as far back as we can go. List names of ALL doctors and cause of death; list ALL disease descriptions as are written, and in the order they are written, establishing which disease is mentioned first. Remember that the disease mentioned first will be the one that goes into the record books. We want to know exactly how many clients have had asbestos-related diseases and then see what they've been classified under.

What age was the deceased at time of death?
Did the deceased spend any time at a hospice? Which one?
Was P.M. carried out on patient? If so by whom and where?
Did deceased receive C.T. scan?
How much time elapsed between biopsy/biopsies and death?
When and where was the biopsy/biopsies performed?
Which surgeon or surgeons were involved?
Was the deceased still entered for a claim? Is there a 'figure'?
Has the surviving spouse or relatives received 'loss of society'?
Had s/he been offered a settlement prior to death?
What was the settlement/settlements?
Who was the action against?
What lawyers were employed? Any other information?

How many cases precisely have gone the whole way through the courts in the past 30 years, whether in England and Wales, or in Scotland? Which company was defendant? Geographical location of workplace? Lawyers involved? M.P. at the period. What was the outcome? Have "exemplary costs" ever been charged against any company? What were the judges' summing up remarks?
Which other companies or bodies do these lawyers act for?
List ALL the medical practitioners involved in individual cases.
Establish any other interests or connections these medical practitioners may have that could have produced a conflict of loyalties.

At which DSS office was deceased registered?
What entitlements had they been receiving?
What time-gap existed between first referral and first allowance?
Has the spouse or any relative of deceased become ill or themselves deceased?
What is the illness description? What was cause of death?

WORK INFORMATION: At which factories/companies/yards or occupations have we established that crocidolite (blue asbestos) was in use.
Which factories/companies/yards or occupations had/have the worst record?
What names do we know of in upper management since the late 1920's.
What trades unions are and have been closely associated? What are the names of the full-time officials at the different periods since the late 1920's. Who were their lawyers? Which insurance or financial institutions were they associated with?

HANSARD: How many questions on asbestos-related topics have been asked in the House of Commons during the past year? The past five years? How many were asked in the period 1948/49/50? How many were asked in 1961/62/63? In 1898/99/1900? In 1906 when the "British Parliamentary Commission were recommending better ventilation and other safety measures"? How many in 1930, 31, 32 following the Home Office report on "widespread asbestos disease in UK factories"? How many in each successive period from any major international finding on asbestos/asbestosis?
Who asked the questions? Which party? Which geographical constituency? Who replied? Which party? geographical constituency?
Can we establish any connection between those who spoke and the the main asbestos-using companies? Connections between shipyards? Connections between insurance companies? Who were the Ministers for Health, Home Office, at the Department of Trade and Industry etc.

INSURANCE: In 1918 Prudential Insurance Co. (USA) produced their "actuarial study showing premature death in the asbestos industry" and the other insurance companies started "increasing premiums and refusing insurance". Which companies were Prudential associated with in Britain? What effects did this study

produce on insurance companies in Britain? (information from available books on subject, plus anywhere else you can think)

Compare the record and manner of industrial operation of Johns Manville and Australia's CSR, with that of Cape, BBA, Turner and Newall and any other appropriate company or corporation. What arguments were being used by the companies when action was brought against them? And any other related question to this or the earlier bits should be added.....

additional medical notes

Lung Function Tests (LFT's):

(Dr B Simpson SMO, BLACKPOOL "When change in airflow occurs in asbestos-related pulmonary disease it is restrictive in nature. An obstructive defect is not a feature. In fact one of the commonest causes of obstructive changes in respiratory function testing in the UK is cigarette smoking."

[*fibrosis*: increase in the formation of fibrous connective tissue either normally as in scar formation, or abnormally to replace normal tissue, especially in the lungs, uterus or heart]

Asbestosis is an inflammatory and fibrotic process of the alveolar structures mediated, at least in part by cytokines released by 'activated' alveolar macrophages. The process of phagocytosis and 'activation' of alveolar macrophages is poorly understood. [NB* In medicine almost anything ending in 'itis' refers to inflammation]

alveolitis is the inflammation of the *alveoli*:

alveoli: tiny saclike structures, especially tiny air sacs of the lungs where the exchange of oxygen and carbon dioxide takes places.

Macrophages: large scavenger cells that engulf and digest invading micro-organisms and cell debris; some are found in connective tissue, liver, spleen; others circulate in blood stream.

cytology: study of the cells.

How do we recognise '*fibrosis*', this disease of the active part of the organ (as opposed to the structural tissue)

NI 226: "fibrosis of the lung parenchyma (*parenchyma* is the active part of an organ as opposed to structural tissue [*stroma*]) is essential for the diagnosis" DSS
DIAGNOSIS:

When there has been a history of substantial exposure to asbestos, a diagnosis of asbestosis can confidently be made if the following findings are present:

(1) the characteristic persistent basal crackles;

(2) radiological features of diffuse interstitial fibrosis in the lower halves of the lung fields;

(3) impairment of lung function consistent with a diagnosis of diffuse interstitial fibrosis.

Lung biopsy undertaken solely for the diagnosis of asbestosis for purposes of compensation is *never* justifiable.

NB. Features which indicate that diffuse interstitial pulmonary fibrosis may have resulted from a condition other than asbestosis are

1) crackles heard all over the chest or predominantly over the upper zones

2) crackles which are coarse in character and occur in expiration as well as inspiration

3) radiological changes consisting of irregular linear opacities in the upper zones as well as the lower zones, especially if they are coarse

4) unexpectedly marked reductions in TLC, FVC, DLCO and KCO for the degree of radiological change

5) rapid development and progression of the disease

Many of these cases will have little or no contact with asbestos.

clinical diagnosis: performed "at bedside" in relation to the patient in contrast to basic-science studies.

histological evidence: relates to the tissue (histology: science of the tissues); slivers of tissue that will reveal evidence of disease

LUNG: either of two large, spongy organs located on each side of the chest cavity, and covered in layers of membrane called the pleura, the outer of which is attached to the rib cage. The lungs constitute the main centres for the exchange of gases involved in respiration. Air breathed into the trachea (the windpipe) is diverted into either of the two bronchi, at which point it reaches a lung. Inside the lung, the air continues down the bronchus, which branches into smaller bronchioles which again branch into tiny alveoli. It is through the thin walls of capillaries surrounding the alveoli that oxygen in the inhaled air is exchanged for carbon dioxide in the

blood, to be exhaled at the next breath. The right lung, with three lobes, is slightly larger than the left, with two, because the left has to make the room for the heart immediately beneath. The short circulation of blood between the heart and the lungs is quite separate from the much longer circulation around the rest of the body.

The doctors up at Blythwood House are *respiratory specialists*; where the tumour is *peritoneal* (as opposed to *pleural*) this is abdominal and the doctors in question are NOT qualified to give specialist judgments, their diagnoses are open to question.

The sorts of statements that you'll find coming from medical authorities on the chest etc. are of the following kind; it helps to clue in on them to defend victims:

Mr So-in-so's "*ischaemic* heart disease accounts for a significant proportion of his breathlessness.

"*Chronic bronchitis* is defined as a cough with phlegm and by itself does not give rise to significant breathlessness unless there is airways obstruction present".

The question is whether the *pleural plaques* are giving rise to his complaint of breathlessness...

"the FEV1 is below predicted and *is often the first indicator of lung restriction*."

"...in addition to pleural plaques there was some *Diffuse Pleural Thickening* which tends to confirm a proportion of the breathlessness being due to the *pleural changes*" (The specialist here recommended a thoracic CT scan before making a final decision).

"The incidence of mesothelioma in the West of Scotland is 40 per 100,000 of the population as compared with 5.7 per 100,000 of the UK pop as a whole (more than 7 times the national average. This high figure is entirely due to the number of men employed in the years in which asbestos was used for lagging. Mr So-in-so's risk is between 1 and 2%"

So how does the latter statistics relate to the 1988 figures of 88% failure in claims on pneumoconiosis at Glasgow? What are the figures on asbestosis? asbestos-related disease generally? Can a prediction on asbestos-related disease generally be made from the mesothelioma cases? Is there a ratio between mes and asb-related? Bring in the 80% asbestos-cancers are lung cancers and only 10% are mesotheliomas.

On asbestos itself:

“Several factors are involved, the most important being fibre size; long thin fibres, usually amphiboles, are the most dangerous. [Why that is] is still controversial. Processing alters fibres and we can recognise those industries in which workers are at less risk, probably due to changes in the fibrous form. Knowledge of which local industries used asbestos in the past can be of great help to a G.P.”

back to diagnosis:

“Rarely necessary *to obtain tissue to make diagnosis* but if necessary then an *open lung biopsy* should be considered. Lung function changes may precede radiological and clinical evidence and, particularly in early stages, it is not uncommon to hear crackles while the chest X-ray is still normal. Finger-clubbing is not an especially helpful factor.